PEASE, (Ed. A.)

VOLUNTARY CONTROL

OF THE HEART.

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VOLUNTARY CONTROL OF THE HEART.[1]

BY EDWARD ALLEN PEASE, A.B. (HARVARD).

ALTHOUGH the heart has always been considered one of the most involuntary organs connected with the human body we have now evidence that there are gifted individuals who have a certain amount of direct control over it. The wonderful connection between the heart's action and a person's feelings is well known. Any feeling of an agreeable or joyful character will tend to increase the rate of the heart-beat, whereas those feelings of a disagreeable or sad nature will tend to decrease it. Moreover, a very strong or sudden emotion of any sort may cause a momentary stopping of the heart-beat, as most people know by experience. Physiologists have long been aware of the close relationship between the heart's action and that of the brain; yet, for lack of sufficient evidence, have not granted that any direct control over the heart could be induced by a simple effort of the will.

In treating the subject of voluntary control of the heart I shall divide it, to avoid confusion, into two parts: 1. Indirect voluntary control, due to the effort of the will directed upon some object or objects which, in their turn, produce an effect upon the heart's action. 2. Direct voluntary control, due simply and purely to an effort of the will directed upon the heart itself.

1. Indirect voluntary control of heart appears in several forms, some of which I will mention with examples, in order that we may have a clear idea of this sort of control before we enter upon the question of direct voluntary control, which forms the special subject of this paper. (*a*) Voluntarily induced sad emotions have been known in several instances to bring about a change in the heart's rhythm. Professor Botkin[2] states he had once a patient under treatment for atrophy of her muscles who could diminish the beat of her heart at pleasure

[1] Read at a physiological conference in the Harvard Medical School, February 4, 1889.

[2] Klinische Wochenschrift. Herausgegeben von Botkin, 1881, No. 10.

and for as long a time as she wished, and, moreover, could destroy its regular rhythm. In order to do this she had only to look at the bed in which she was formerly ill and thus bring into her mind the unpleasant associations surrounding it. When in good spirits she had no control over her heart. (*b*) Pressure on the vagus (a large nerve running alongside the carotid artery in the neck, and, on its way to the stomach, sending inhibitory branches to the heart), has been brought into service to cause a decrease in number of heart-beats. Professor Czermak,[3] of Prague, could diminish the rate of his heart-beat more than one-half by simply pressing on this nerve. As he had an enlarged indurated lymph gland lying just beneath the vagus nerve in the upper portion of his neck he found no difficulty in bringing about this pressure. (*c*) Checking the respiratory rhythm by a strong contraction of the thoracic muscles while the glottis is closed in inspiratory position has been sufficient to stop the heart-beat. Weber[4] has performed this experiment successfully on himself, and explains this decrease in the rate and stoppage of the heart's beat, partly by a lessening of the pressure within the thorax and partly by a storing up of carbonic dioxide gas by means of which the inhibitory heart-centres going to the medulla oblongata become irritated. But Wertheimer and Meyer[5] explain a similar case by saying the diminution in the number of beats was due to irritation of the threads of the vagi which run to the lungs. These threads become pressed together by the full alveoli and so are stimulated. (*d*) A marked change made in the respiratory rhythm will cause a change in the rapidity of the heart's beat; but an increased number of heart-beats from this cause would really come under the head of increased heart-action due to increased muscular action of any sort, — a phenomenon with which we are all familiar.

2. Direct voluntary control of heart. Carpenter[6] says that the influence of the state of expectant attention is strangely manifested in the heart; the action of which, Sir H. Holland has remarked, "is often quickened or otherwise disturbed by a mere centering of consciousness upon it without any

[3] Jenaische Zeitschrift f. Med. und Naturwiss., 1865, p. 384.
[4] Weber. Ueber ein Verfahren, den Kreislauf des Blutes und die Function des Herzens willkürlich zu unterbrechen. Arch. f. Anat. und Physiol. und wissenschaftl. Medicin, von T. Müller, 1851, p. 88.
[5] Archives de Physiologie, Paris, 1869, p. 49.
[6] Human Physiology, Carpenter, 1869, p. 808.

emotion or anxiety; and where there is a liability to irregular pulsation such action is seemingly brought on, or increased, by the effort of the attention, even though no obvious emotion be present." We know that the vagus nerve has inhibitory and the sympathetic nerve accelerator action upon the heart. A voluntary control apparently from the action of one or the other of these nerves can thus be effected by certain persons who possess the requisite amount of will-power.

Joseph Frank[7] made his heart's beat intermittent and irregular by simply concentrating his attention upon it. A Bologna professor[8] was completely cured of intermittent pulse by following the advice of his physician, Morgagni, not to count his pulse, *i.e.*, not to pay any more attention to it. There was a Fellow of the Royal Society in London[9] who could increase his heart's beat ten to twenty times a minute by a simple effort of the will.

The best known and most historic case of voluntary control of heart is that of Lieutenant Townsend.[10] He possessed the power to stop at will his heart-beat and breathing, and at the same time to go into a deathlike sleep. His body would begin to cool and stiffen, his eyes become immovable, and finally his consciousness almost leave him. In a few hours, however, he would return to full consciousness. One evening, after he had made an experiment before a large audience to show his power of voluntary control of heart, this man died. From the fact that Lieutenant Townsend died soon after the only experiment of which we have an accurate description it might be thought that he was already in a moribund condition before trying this experiment; yet the post-mortem examination revealed a diseased condition in no part of the body except one kidney. In all the cases I have mentioned so far there is no conclusive evidence that the change in the heart's rhythm was due simply to direct voluntary control. To prove this all forms of indirect voluntary control must be excluded.

It was by the aid of the sphygmograph, pneumograph, plethysmograph, and other instruments that Professor Tarchanoff,[11] a Russian physiologist, was

[7] Joseph Frank. Praxæ medicæ universæ præcepta. Lipsiæ. Th. ii. Bd. ii. Abth. ii, p. 373.
[8] Wagner's Handwörterbuch d. Physiol., 1844, Bd. ii. p. 82.
[9] D. H. Tuke. Illustrations of the Influence of the Mind upon the Body in Health and Disease, London, 1872.
[10] Carpenter's Human Physiology, 1853, p. 1103.
[11] Ueber die Willkürliche Acceleration der Herzschläge beim Menschen. Archiv für die Gesammte Physiologie, Bd. 35, p. 109.

able to prove scientifically that in certain cases the heart is under the direct control of the will.[12] He found among his students a young man named Salomé, who claimed to have voluntary control over his heart. The young man was of a rather nervous temperament, and, when ten or fifteen years of age, had suffered from palpitation of the heart without any apparent cause. In course of time, however, he had rid himself of the disease and would have forgotten all about it, were it not for the fact that he noticed accidentally he was able to change the rhythm of his pulse by a comparatively trifling outside cause. Experimenting further he found he could accelerate his pulse by simply concentrating his attention upon it and exerting a sufficient amount of will-power. It was at this time that Salomé came under the attention of Professor Tarchanoff.

At the first meeting Salomé gave a striking proof of his ability by accelerating the beat of his heart from 70 to 105, *i.e.*, 35 beats a minute. The experiment was repeated several times with about the same result, although the amount of the acceleration fell off plainly with each repetition of the act. In later experiments Salomé increased the beat somewhat more. He found that arsenic would assist him in accelerating the heart's beat, and that nitrous oxide (laughing gas), although increasing the heart-beat 6 or 8 a minute by its own properties, would at the same time take away the power of the subject to control his heart voluntarily. When he recovered from the effects of the nitrous oxide he was again able to accelerate the beat 25 a minute. Salomé said he called up no extraneous ideas to increase his heart's beat, but used a special effort of the will. Throughout the experiments Tarchanoff found that the respiration during acceleration was not quite normal, as may be seen by inspection of the pneumographic tracing marked 3 in Fig. I., a reproduction of one of Tarchanoff's figures. Tracing 2 shows the period of acceleration by a heavy white line made by the subject pressing gently an electric button during the time he was trying to increase his heart-beat. Tracing 1 is made by a galvanic chronograph marking seconds. Tracing 4 was obtained by placing the foot in a plethysmograph, a rubber cap arrangement fitting tightly over the leg and having the air space between the

[12] I am indebted to Professor Tarchanoff's article for several of my references.

foot and the cap connected with a Marey drum. It shows the increased heart-beat during the acceleratory effort and the diminution in the size of the limb as appears from the general falling of the line. This diminution is associated with an increased blood-pressure, which lasts after the effort at acceleration has ceased, thus showing that a vaso-constrictor influence accompanies voluntary acceleration.

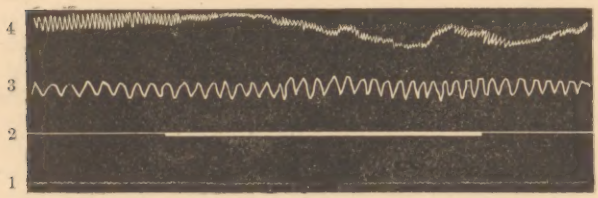

FIG. I. (from Professor Tarchanoff's article). — 1. Chronograph, one-second interval. 2. Period of acceleration. 3. Thoracic pneumograph. 4. Plethysmograph. To be read from left to right.

The acceleration of the beat in the above figure is at the rate of 27 a minute. There is also an increase of three respiratory curves a minute; but this slight increase in the number of respirations is quite insufficient to account for the great increase in the number of heart-beats. Salomé's vaso-motor system was very sensitive: even slight sounds in the room would increase his blood-pressure. He had control over his ear muscles, platysma myoides, and several other muscles which are not usually under the control of the will. Tarchanoff thinks that Salomé gets his power of voluntary control from an unusual organization of the nervous system. He believes there is a direct connection between the highest will centres of the hemispheres and the accelerating heart centres, which are situated in the upper cervical part of the spinal cord. He thinks the vagus plays no part in this acceleration. Tarchanoff says that after these experiments on Salomé he met several persons who had the power to accelerate their heart's beat; but all, without exception, had also a certain amount of control over muscles which were ordinarily beyond the voluntary control of man, such as the platysma myoides, muscles of the ear, and of the three terminal phalanges of the fingers. He had not met a single person having the usual control of the muscles who could influence his heart's beat.

By chance I heard that a member of the Harvard Medical School had a certain amount of voluntary control over his heart. This man, on learning I had chosen the subject of "Voluntary Control of Heart" to write on, offered to allow me to try some experiments upon him to test scientifically his power. As I knew practically nothing about the delicate apparatus used in experiments of this sort, Dr. H. P. Bowditch kindly conducted the experiments, the results of which I now present to you.

In these experiments a sphygmograph, pneumograph, chronograph marking two-second intervals, and an instrument to mark the period of acceleration, were used. Our subject, like Salomé, was unable to continue the stimulus for acceleration of the heart indefinitely, so in the following experiments I have reduced the actual figures to the basis of a minute. As the thoracic respiratory curve in our first experiments, as shown by the pneumograph, was rather small, a second pneumograph was placed over the subject's abdomen. The respiratory curve produced by this last instrument was much better marked. By means of these two

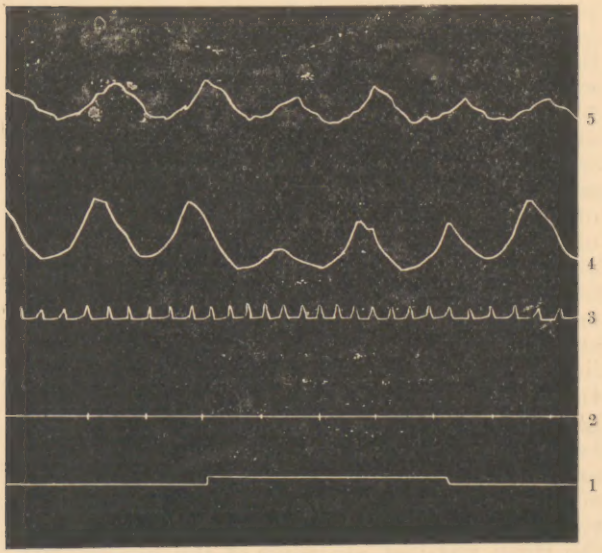

FIG. II.—1. Period of acceleration. 2. Chronograph, two-second intervals. 3. Sphygmograph on radial artery. 4. Abdominal pneumograph. 5. Thoracic pneumograph. To be read from right to left. An acceleration at the rate of 17 beats per minute.

instruments we could detect in the remaining experiments any slight change in the breathing.

Now the first figure I have for your inspection is one showing a great acceleration of the heart's beat with a fairly normal respiration. (Fig. ii. p. 6.)

This figure and also the following figures should be read from right to left. Tracing 1 marks the acceleration period by making a somewhat higher line during the effort. Tracing 2 is made by a chronograph marking two-second intervals. Tracing 3 is that of the transmission sphygmograph placed upon the radial artery of the right hand. Tracing 4 is that of the abdominal pneumograph. Tracing 5 is that of the thoracic pneumograph. The descending curves in the last two mark inspiration; the ascending curves, expiration. Now, in order to find out the acceleration I counted the heart-beats on the sphygmographic tracing between perpendicular lines drawn from the chronographic dots nearest the beginning and end of the acceleration period (having made corrections for any lack in perpendicularity there might have been in the position of the tracing pens), and then subtracted from the result thus obtained the number of heart-beats in the same length of time preceding the point where the stimulus for acceleration was put on. For the 8 seconds *before* acceleration there were 10.4 heart-beats. For the 8 seconds *during* acceleration there were 12.7 heart-beats. The acceleration in this case was 2.3 beats in 8 seconds, which is at the rate of 17¼ beats in one minute. The beat after acceleration does not return immediately, but gradually, to normal; as I found by counting the number of beats for the same length of time after the accelleratory effort had ceased to be called into action. Throughout the experiments I found that each acceleration was attended with a somewhat deeper inspiration, followed by a shortened expiration. This may be seen in tracing 4 in Fig. II. and to a less degree by tracing 5. The breathing becomes a trifle quicker during acceleration, but the increase is so slight that it is barely noticeable in the tracing. The question is whether the slight increase in rapidity and depth of respiration would cause an acceleration at the rate of over 17 beats a minute. This is not probable, as I will show later.

Fig. III. shows the result of an experiment in which the subject tried to accelerate his heart-beat as much as possible, without, however, any powerful

muscular contraction. (In the preceding experiments he had tried to breathe normally while accelerating.) The acceleration obtained was at the

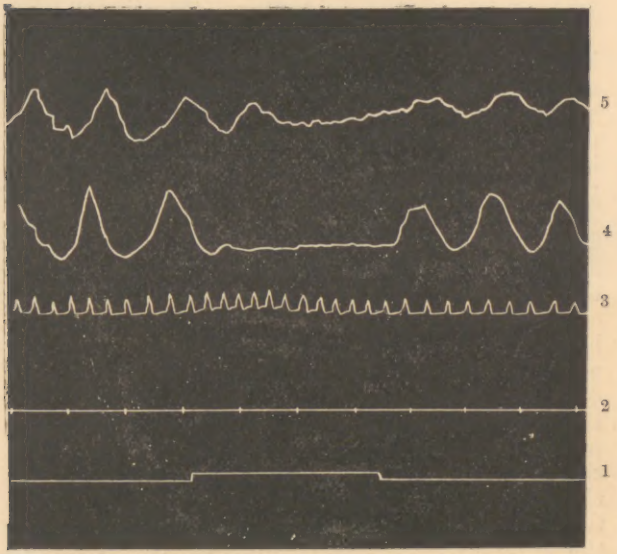

FIG. III. — 1. Period of acceleration. 2. Chronograph, two-second intervals. 3. Sphygmograph on radial artery. 4. Abdominal pneumograph. 5. Thoracic pneumograph. To be read from right to left. Acceleration at the rate of 27 per minute.

rate of 27 a minute, the beat going from 79 to 106.

In order to get this great acceleration he held his breath at the middle of the thoracic respiration (as may be seen by referring to the figure), and then called up the stimulus to accelerate. It is to be noticed in this figure that there is a distinct rise of blood-pressure accompanying the acceleration of the heart-beat as seen by the general rise of the sphygmographic tracing. The increased blood-pressure appears soon after the effort at acceleration has begun, and lasts for some little time after the effort has ceased. The rise is better marked here than in the preceding or following figure. From the fact that his respiration stopped when the acceleration was most marked, it seemed possible that holding his breath might be the main cause of the great acceleration. Therefore an experiment was tried where the subject held his respiration until a tracing of the pulse was obtained when the heart was

under this condition, and then the word was given to accelerate.

The result of this experiment is seen in Fig. IV. The period of acceleration was six seconds. The normal beat was at the rate of 77 a minute; with checked respiration the beat was at the rate of 73 a minute; during acceleration the beat was at the rate of 87 a minute, — an acceleration, after the diminution caused by checked respiration, at the rate of 14 a minute. In another experiment similar to the one above the subject caused a still greater acceleration of his heart-beat, the sphygmograph registering an increase at the rate of 19½ beats a minute. These facts eliminate the possibility that

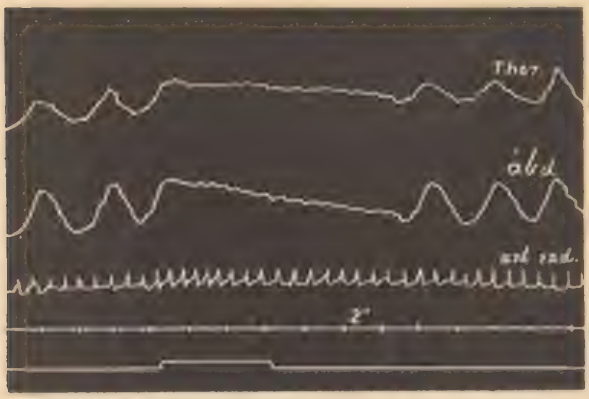

FIG. IV. — 1. Period of acceleration. 2. Chronograph, two-second intervals. 3. Sphygmograph on radial artery. 4. Abdominal pneumograph. 5. Thoracic pneumograph. To be read from right to left. An acceleration of 14 per minute.

the acceleration was in any way due to a checking of the respiration, for, in this subject, holding the breath caused a very marked decrease instead of an increase in the number of heart-beats per minute.

In speaking of Fig. II. in these experiments I left for later consideration the fact that, although the subject endeavored to keep his breathing normal, there was with each acceleration a slightly deeper and quicker respiration. Now, in order to prove that the acceleration was not due to any increase in frequency or depth of breathing, the subject was asked to take several deep and rather quick respirations, without putting on the acceleration stimulus. In comparing the tracings of sev-

eral periods of normal respirations with several periods of these deeper and rather more rapid respirations I found that the moderately fast and deep breathing caused but very little increase in the rate of the heart-beat, — an increase that was totally insufficient to be considered the cause of the great acceleration obtained by this subject.

In the above remarks I have taken care to say periods of respiration (meaning thereby a certain number of full respirations), for Wertheimer and Meyer [13] say there is an acceleration of the heart-beat at each inspiration and a diminution at each expiration. As there were one or more full respirations in the cases I have presented to you my results are not dependent on any acceleration that may be found in a single inspiration. While bringing the Fig. II. to your attention I asked you to notice the marked rise in blood-pressure during acceleration as shown by the general rise of the sphygmographic tracing. The cause of this rise in blood-pressure is shown in the plethysmographic tracing in Fig. V. The plethysmograph used for this tracing was a small rubber tube fitting closely over the third finger and having between it and the end of the finger an air space delicately connected with a Marey drum.

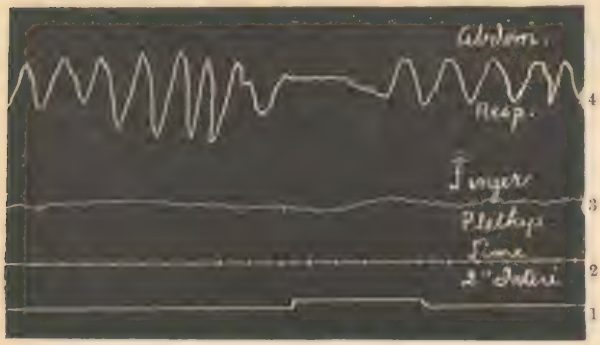

FIG. V.—1. Acceleration period. 2. Chronograph, two-second intervals. 3. Finger plethysmograph, on third finger. 4. Abdominal respiration. To be read from right to left.

The general fall in the plethysmographic tracing, appearing soon after the acceleratory stimulus, is caused by the diminution in size of the extremity,

[13] Archives de Physiologie, p. 24.

and indicates an increased blood-pressure due to a peripheral vaso-constrictor influence, accompanying the increased heart-beat. This result accords with Foster's[14] general statement that "variations in the heart-beat must always be looked upon as a far less important factor of blood-pressure than variations in the peripheral resistance." The tracing marked the greatest contraction of the organ, *i.e.*, the highest blood-pressure, very near the end of the acceleration period. There was then a secondary rise in the tracing similar to the one which Tarchanoff got in his experiments on Salomé.

Foster [15] also says that high blood-pressure is attended, contrary to expectation, by a slow heart-beat; and Marey states that the rate of the heart-beat is in inverse proportion to the arterial pressure, a rise in pressure being accompanied by a diminution in the pulse-rate. This fact is shown in the sphygmographic tracing on Fig. IV., where a slight rise in blood-pressure appears on holding the breath, and there also appears a corresponding decrease in the number of heart-beats until the stimulus for acceleration was put on, as I said while speaking of that figure. Since, then, a decrease in the number of heart-beats appeared with the rise in blood-pressure, it must be admitted that increased blood-pressure could not have been instrumental in bringing about this acceleration of the heart.

It is well to state here, perhaps, that the subject is a healthy, vigorous man who has never had any trouble with his heart. He has a certain amount of control over his ear muscles and platysma myoides. With each effort at acceleration of the heart he feels a sort of tingling reaching from the cerebral region to the heart and then spreading out toward the periphery. The accelerating impulse is not a single continued one but a series of short efforts gradually growing weaker. He could accelerate most when he felt strongest. His accelerating power decreases with every experiment, until finally he feels himself quite unable to produce further acceleration. After each experiment the perspiration would stand out in great drops on the palms of his hands, thus showing an intimate relation in the medulla oblongata between the cardiac nerves and the vaso-motor system. A slight head-

[14] Foster's Physiology, Third American Edition, Reichert, 1885, p. 242.
[15] *Loc. cit.*, p. 240.

ache followed the experiments, if they were somewhat prolonged. The subject is apparently unable to decrease in the least the beat of his heart.

From what I have said and shown to you I have come to the conclusion that the power of this subject to accelerate his heart-beat was not due to the calling up to his mind of any emotions or ideas which had a tendency to change his heart-beat (the word of the subject must of necessity be taken for this, as no experiment can prove it), nor to any movement of the regular voluntary muscles, nor to a change in breathing or blood-pressure, but was due simply and purely to an effort of the will directed upon the regulating mechanism of the heart.[16]

I here append, as a caution to those who are tempted to investigate for themselves this interesting subject, a translation of a letter which Prof. Tarchanoff wrote to the editor of *Die Archives für die Gesammte Physiologie* a short time after the appearance of his article: —

"It was very pleasant for me to learn that my work on the voluntary acceleration of the heart in man has been of such lively interest to you. This peculiar power is indeed remarkable, but unfortunately is very dangerous for those persons who have this gift, as I was unfortunately convinced in the case of Dr. Schlesinger. He could even double voluntarily the number of his heart-beats. But he now suffers from such a severe palpitation of the heart that he can no longer sleep quietly. Since I fear that the experiments made by me with Dr. Schlesinger may have caused these evil effects, I would ask other investigators to exercise the greatest caution in repeating my experiments.

[16] The figures used in this article are reproductions of photographs taken from the original tracings.